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OBSERVATIONS ON THE STATE OF THE VASCULAR SYSTEM AFTER DEATH BY ASPHYXIA AND BY CARDIAC FAILURE.*

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Asphyxia.

THE characteristic distribution of the blood in the heart and great vessels after death by asphyxia has been ascribed to various causes, and a glance at the textbooks now in use will show that different opinions are still held as to why, on examination of the body some hours after death, the right side of the heart and the great veins opening into it are usually found to be largely distended with blood, while the left heart is empty or contains little blood.

Among various causes that have been assigned there may be mentioned as the chief ones: (1) The greater distensibility of the right heart; (2) the influence of violent respiratory movements and muscular spasms in drawing and driving blood into the great veins and right heart; (3) an alleged strong contraction of the pulmonary arterioles opposing the discharge of blood from the right ventricle, and so leading to an accumulation of blood in the right heart and great veins and a state of comparative emptiness on the left side; (4) the influence of rigor mortis coming on in the left ventricle after death.

Before entering on a brief consideration of these causes, it is important to determine the actual condition of the heart at the moment of death. This is a subject on which there is still a certain amount of contradictory assertion, though there is really no room for doubt as to the facts. It is quite certain that when death occurs from asphyxia the whole heart stops distended with blood—both auricles and both ventricles. This is the case whether asphyxia has been caused by occluding the trachea during natural breathing (in an anaesthetized animal), and then opening the thorax and examining the heart when death has occurred; or whether the thorax has first been opened—artificial inflation of the lungs being per-

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formed—and then asphyxia induced by stoppage of the artificial inflation. In the latter case the condition of the heart may be watched during the whole process of asphyxia; it is seen (as has often been described) that in the earlier phase of asphyxia when the great rise of arterial blood pressure takes place, the left heart becomes greatly distended in consequence of the inability of the left ventricle to empty itself against the enormously increased peripheral resistance caused by the contracted state of the small arteries. Distension of the right heart speedily supervenes—mainly dependent on the backward pressure arising from the inability of the left heart to pump on its blood efficiently. The whole organ thus becomes greatly distended in all its parts, and when after a time it ceases to beat both sides are still distended—though often not quite so largely over-filled as during the convulsive stage.[1]

Blood Pressure on the Right and Left Sides of the Heart immediately after Death.

As there seems to have been no determination of the condition of the right and left chambers as regards the pressure in their interior when death occurs, I have made a number of observations on this important point.

1. By introducing a cannula into the right and left sides respectively, and connecting the cannula with a mercurial manometer in the usual way.

2. By introducing the point of a vertical glass tube of small calibre into the right and left chambers, and observing the height to which the blood rose in each case, precautions being taken as to exact levels, capillarity, etc.

By these methods I have found equal pressures on the right and left sides, a slight positive pressure of a few millimetres of mercury (commonly about 3 mm. in the cat and rabbit) being constantly present. Thus it is evident that differences in the state of the two sides cannot be accounted for by differences of pressure.

As regards the relative amounts of blood on the right and left sides various conflicting statements have been made, one observer describing a preponderance on the left side as compared with the right (in the proportion of 3 : 2), while another finds as the result of an experiment a great preponderance on the right side—in the ratio of 16 : $2\frac{1}{2}$.

The method of determination adopted has been to ligature the great vessels, taking care to close the coronary arteries, and then evacuate the contents of the right and left sides separately and measure them. I have always found a large amount of blood on both sides, but always a greater amount on the right side; this is evidently due to the greater distensibility of the right heart, since the pressure on the two sides is equal.

Remarks on Alleged Causes of the Characteristic Distribution of the Blood.

1. The greater distensibility of the right heart, while apparently the cause of there being more blood on that side, would by no means account for the left ventricle being empty or nearly empty. Further, the greater distensibility of the right side is present in death from other causes—syncope, etc., so that this factor could not account for the characteristic asphyxial condition recognized at the necropsy.

2. The influence of respiratory movements and muscular spasms in leading to an accumulation of blood on the right side as has been affirmed, is clearly not a necessary condition, since the state of the heart does not essentially differ whether (*a*) those movements have been prominent during asphyxia, or (*b*) have not occurred at all in consequence of a depressed condition of the medulla, administration of curare, etc.

3. The hypothesis that extreme constriction of the pulmonary arterioles arrests the circulation and is the immediate cause of death in asphyxia has been strongly advocated by the late Sir George Johnson.[2]

But this view is entirely negatived by a variety of evidence.

(*a*) Direct observation of the heart during asphyxia or immediately after death. As above stated the whole organ becomes greatly distended during asphyxia and is still largely distended on both sides when death occurs — a condition which persists for some time after death—this period varying in different circumstances. Johnson on the other hand describes the left heart as becoming collapsed and empty (or almost empty) during the latter phase of asphyxia—on account of an arrest of the circulation through the lungs. It seems to me that an early development of rigor mortis in the left heart may have been concerned in producing such appearances. For rigor sometimes does begin very early in the left ventricle; I have seen the left ventricle contracting in incipient rigor as early as nine minutes after closure of the trachea in the rat, twelve minutes in the rabbit, and fifteen minutes in the cat, though the time is, as a rule, much longer than the periods stated.

(*b*) There is a considerable pressure of blood in the peripheral end of a pulmonary vein at any stage of asphyxia—transmitted from the pulmonary artery, through the capillaries and showing that the blood flow is not arrested in the lung.

(*c*) The blood pressure in the left auricle during asphyxia does not show any indication of an arrest of the circulation in the lung.[3]

(*d*) The hypothesis that extreme constriction of the pulmonary arterioles is excited by asphyxial blood acting through the vasomotor nerves can be tested by a form of experiment in which the vasomotor effects of asphyxia can be produced apart from the complicating action of asphyxial blood upon the heart itself. This can be done by closing the arteries to the head (carotids and vertebrals), and so producing an asphyxial condition in the brain, medulla, and upper portion of the spinal cord. The usual exaggerated respiratory movements and convulsive spasms of asphyxia speedily develop, and after passing through the usual stages all these movements cease. Meanwhile the great asphyxial rise of blood pressure has taken place, in consequence of the excitation of the vasomotor centre by asphyxial blood, and the whole heart becomes distended in the usual fashion. But if the blood circulating through the heart (and body, excluding the head) is prevented from becoming asphyxial by artificial inflation of the lungs, the circulation does not become arrested, in spite of the vasomotor spasm excited as usual by the asphyxial condition of the medulla, etc. On the contrary, the heart goes on beating, and in a few minutes the vasomotor spasm passes off—with the death of the vasomotor centre—the excessive blood pressure falls, the over-distended

heart is relieved, and if means are taken (pressure on abdomen, etc.) to maintain a fair blood pressure by compensating for the loss of the normal vasomotor tone, the heart action and circulation go on well for many hours. Here it is evident that extreme vasomotor spasm does not arrest the circulation as long as the heart is supplied with arterialized blood; the essential cause of failure in ordinary asphyxia is plainly due to the asphyxial character of the blood supply to the heart itself.

(*e*) The circulatory phenomena in asphyxia are quite different from what is seen when the pulmonary vessels are actually obstructed. The injection of lycopodium spores into the jugular vein so as to plug the pulmonary vessels is a method that I have adopted in order to study the effects of undoubted pulmonary obstruction. When this is done, the respiratory movements speedily assume the asphyxial character, convulsive contraction occurs in the skeletal muscles with constriction of the systemic arteries due to vasomotor spasm, etc. But the changes in the heart and the pulmonary circulation are very different from those seen in ordinary asphyxia. The pulmonary artery quickly swells up in consequence of the excessive pressure in its interior due to the obstruction in the small vessels; the right ventricle and auricle become over distended, while the left auricle and ventricle become small and look almost empty—as immediate consequences of the pulmonary obstruction. (Some time later the left ventricle comes to contain a good deal of blood, while the left auricle remains very small).

Changes in the Heart after Death.

The characteristic asphyxial condition of the heart as seen some hours after death is a *post-mortem* development, due to rigor mortis in the cardiac muscle.

With regard to the causation of the change which occurs, certain prominent features in the process of asphyxia seen prior to death are apparently unessential — for example, the great rise in systemic pressure and consequent strain upon the cardiac muscle; for when the usual rise of pressure fails to occur (from excessive dose of curare, etc.) the asphyxial distribution of the blood may still be found. Thus on weighing the clots obtained from the two sides in such a case, the right ventricle clot was 1,120 mg. (in a rabbit), while the left one was 490.

Graphic Records of the Expulsive Action of the Left Ventricle in Rigor.

In studying the influence of rigor in emptying the left ventricle I have obtained tracings of its expulsive action by tying a cannula into the aortic orifice of a heart immediately after death, and connecting the cannula in the usual way with a mercurial manometer recording upon a slow drum. The rise of pressure caused by the contraction of the ventricle as it enters into rigor was often about 30 mm. Hg. in the cat, and in some instances curious features were evident in the form of slow secondary oscillations, indicating minor contractions and relaxations of the ventricular wall.[4]

It is evident that the expulsive power of a strongly-marked rigor contraction is much more than would be necessary to expel its contents against the very low resistance present after death—a pressure in the aorta of a very few millimetres

of mercury. When the state of the heart is watched for some time after death, the gradual contraction and the expulsion of the ventricular contents may be directly observed, together with the development of the other signs of rigor—increased firmness of the muscle, change in colour, etc.

Why is the Right Ventricle not Emptied?

After asphyxia the right side of the heart dies slowly and gradually, much later than the left side. Rigor in the right ventricle has very little expulsive power on account of its lateness in development, its gradual onset, and unequal distribution. As a muscle exerts no contractile pull after rigor has been established, but only between the commencement of rigor and its establishment, it is evident that, if the period of attempted shortening does not coincide in the various fibres little result can be produced.

Variations in Time of Onset and Strength of Rigor.

Lateness in the appearance of rigor is unfavourable to the expulsion of blood from the cardiac chambers; for the resistance increases on account of changes in the blood, increased adhesiveness, occurrence of coagulation, etc.

In some instances of death by asphyxia, where rigor in the heart has come on early and been strongly marked (rabbit and rat) I have found *both* ventricles empty after death.

In some other cases again, where rigor has been feeble even in the left ventricle, I have found that the usual distribution of blood was not present, both ventricles containing much blood.

When death is caused by the gradual inhalation of weak chloroform vapour, and the respiration has stopped before the heart beat, the usual asphyxial condition is found in the heart and great vessels after death.

Cardiac Failure, etc.

When death is caused by primary cardiac failure, prior to the cessation of respiration, the heart usually stops beating distended with blood in all its parts. Immediately after death the blood pressure in the right and left chambers is found to be equal; the amount of blood is greater in the more distensible right heart, though there is much blood in the left heart also.

Important changes commonly occur in the condition of the organ and the distribution of the blood after death.

Chloroform Syncope.

Cardiac failure, while the respiration went on for some time, has been induced in several animals (cats) by the rapid inhalation of chloroform during quick deep respiration. The thorax being opened as quickly as possible after cardiac collapse had occurred and while respiratory movements were still going on, the whole heart was found to be largely distended and showing only feeble and entirely ineffective attempts at contraction.

Some hours after death the left heart was rigid and small, while the right side was still distended, that is, an asphyxial distribution of the blood was present. Hence such a *post-mortem* condition is no indication as to whether death had been initiated by cardiac or by respiratory failure.

Results similar to the preceding were obtained when cardiac paralysis was caused by means of chloroform vapour added to the air used in artificial inflation of the lungs, instead of natural inhalation. The heart may in this way be rapidly paralysed.

In some instances both ventricles were found rigid and nearly empty after the heart had been paralysed by chloroform; in some others both ventricles contained a good deal of blood a number of hours after death. It would obviously be a mistake to expect that a heart paralysed by chloroform should be found in a relaxed and distended condition on examination some hours *post mortem*.

Ammonia and Lactic Acid.

Either of these substances injected into the jugular vein quickly paralyses the heart in the distended condition; the respiration soon stops. Necropsy some hours later shows the condition of the heart and great vessels usually associated with asphyxia.

Sodium Carbonate and Morphine Acetate.

When cardiac failure is caused by injection of either of these into the jugular vein, the heart, stopping in the usual state of distension, changes very strikingly soon after death; both ventricles contract and become practically empty; the left auricle also contracts and contains little blood.

Potassium Salts.

The heart poisoned by an overdose of a potassium salt injected into the circulation commonly remains more or less distended after death. On examination twenty-four hours *post mortem*, a large amount of blood (clots) is usually found on both sides of the heart, in the left ventricle as well as in the right ventricle.

From the foregoing observations it is evident that after cardiac failure the *post-mortem* condition found some hours later varies widely according to the means used to paralyse the heart: (1) The condition usually associated with asphyxia may be present; or (2) both ventricles may be contracted and practically empty while the left auricle contains little blood; or (3) the heart may contain much blood on both sides.

Death from Haemorrhage.—After death both ventricles go into marked rigor, and are found contracted and almost empty. Left auricle very small. Right auricle contains a fair amount of blood.

Death from Embolism of the Pulmonary Vessels.

Post mortem, the right heart is found to be distended, the left ventricle contracted and nearly empty, left auricle small.

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NOTE AND REFERENCES.

1 All experiments referred to in this paper were performed on anaesthetized animals. 2 *An Essay on Asphyxia*, London, 1899. 3 *Cf.* Martin, *Proc. Roy. Soc.*, xlix, p. 145; Bradford and Dean, *Journ. of Physiol.*, xvi, p. 74. 4 *Journ. of Physiol.*, xxvii.